modulating the differential response to FGF. Founder cells exhibit an enrichment of membrane protrusions on their anterio-ventral surface, where the heart daughter will arise. We have shown that delocalized, constitutive activity of Cdc42, a Rho GTPase responsible for the formation of filopodia in many systems, disrupts the polarity of founder cell division. Founder cells expressing delocalized, constitutively active Cdc42 have membrane protrusions placed uniformly around the cell, and both daughters take on heart fate. Polarity can be restored by blocking Cdc42-directed actin polymerization using a truncated form of WASP. To further characterize the relationship between protrusive activity of the actin cytoskeleton and FGF signaling, we will employ live fluorescence microscopy to observe the localization and movement of several components of the signaling pathway downstream of FGF. These results highlight a potential role for the cytoskeleton in regulating differential fate induction events during development.

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Program/Abstract # 312
A feedback loop between xylt1 and sox9 controls chondrocyte differentiation

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Cells must interact with their extracellular environment as they differentiate. Here, we use zebrafish mutant embryos to reveal complex interactions between proteoglycans and chondrocyte differentiation that may regulate the timing of skeletal development *in vivo*. The enzyme Xylosyltransferase1 (Xylt1) catalyzes proteoglycan synthesis, and the transcription factor Sox9 drives chondrocyte differentiation. We find that *xylt1* expression is restricted spatially to differentiating chondrocytes, which express both zebrafish *sox9* co-orthologues. Supporting the hypothesis that Sox9 regulates proteoglycan synthesis genes, *xylt1* expression appears reduced or absent in *sox9* mutant chondrocytes. On the other hand, expression of *sox9* genes is down-regulated in *xylt1* mutants. These changes appear only after chondrocytes of wild-type siblings begin to secrete abundant proteoglycans, suggesting a positive feedback onto chondrocyte differentiation from their extracellular environment. Interestingly, this positive feedback loop between *xylt1* and *sox9* appears to control the timing of chondrocyte differentiation: *xylt1* mutant chondrocytes express markers of chondrocyte maturation, such as *collagen type 10a1* and *indian hedgehog (ihh)* co-orthologues, earlier than wild-type siblings. Ihh is known to induce bone in the perichondrium, a tissue that overlies developing chondrocytes, and *xylt1* mutants have early perichondral bone. Consistent with the idea that early *ihh* expression in *xylt1* mutant chondrocytes causes precocious perichondral bone, no early bone forms in *xylt1;ihha* double mutants.

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Program/Abstract # 313
Identification of a novel protein, LRRP, involved in primitive erythropoiesis and non-canonical Wnt signaling

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In *Xenopus*, signals from the ectoderm are required to induce mesoderm to adopt an erythroid fate. Our prior work demonstrated that the transcription factor GATA-2 is essential for this inductive signal. Using microarray analysis, we have identified a gene encoding a novel leucine-rich repeat protein (LRRP) that is upregulated by GATA-2 in the ectoderm, and required for erythropoiesis. Our microarray data also indicate that members of the *non-canonical* Wnt pathway are upregulated by GATA-2, whereas genes associated with the *canonical* Wnt pathway are downregulated by GATA-2. Reciprocal regulation of these two pathways by GATA-2 is intriguing given recent evidence that GATA proteins are involved in promoting the switch between canonical and non-canonical Wnt signaling in other developmental contexts. Moreover, coordinate regulation of these two often opposing pathways may be required for the transition between progenitor expansion, and differentiation to more mature cell types. Interestingly, in addition to its function during erythropoiesis, LRRP appears to be both necessary and sufficient to activate non-canonical Wnt signaling during convergent extension and heart development. We predict that non-canonical Wnt signaling is activated downstream of GATA-2, possibly in an LRRP-mediated fashion. We further hypothesize that these events are required to inhibit canonical Wnt signaling to allow blood progenitors to exit the cell cycle and adopt a hematopoietic fate.

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Program/Abstract # 314
Tbx6-dependent regulation of Sox2 enhancer N1 determines the neural vs. mesodermal fate of axial stem cells in the caudal lateral epiblast

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Recent cell lineage analyses indicate that the region of epiblast flanking the rostral primitive streak (Caudal Lateral Epiblast, CLE) serves as the bipotential precursor for both the neural plate and paraxial mesoderm. However, regulatory mechanisms on how each cell acquires its fate from bi-potent progenitor state and establishes its trait remain unknown. We demonstrate that Tbx6-dependent regulation of Sox2 enhancer N1 determines the neural vs. mesodermal fate. In wild-type embryos, enhancer N1 of the pan-neural primordial gene Sox2 is activated in the CLE to drive Sox2 expression in the neural plate, but it is turned off in the mesodermal precursors formed after ingression through the primitive streak. In Tbx6 mutant embryos, however, enhancer N1 activity persists in the paraxial mesoderm compartment, eliciting ectopic Sox2 activation and transforming the paraxial mesoderm into ectopic neural tubes. Targeted deletion of enhancer N-1 in Tbx6 mutant embryos caused loss of paraxial tube formation as well as loss of ectopic *Sox2* expression, indicating that SOX2 is a key mediator in the formation of the ectopic neural tube. Our results suggest that neural/mesodermal fate choice operates in the mesodermal precursors derived from the CLE, and that Tbx6-dependent repression of Sox2 enhancer N1 is required to specify paraxial mesoderm.

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Program/Abstract # 315
Transcriptional control of dorsal-ventral polarity cues in C. elegans

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Embryos must undergo a series of asymmetric divisions to establish the three main axes of the body (anterior–posterior, left–